EFFECT OF CONCENTRATE FORM ON GASTRIC ULCER SYNDROME IN HORSES

A Thesis

by

LINDSEY RAE HUTH

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

December 2011

Major Subject: Animal Science

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Approved by:

Co-Chairs of Committee,	Dennis H. Sigler
	Clay A. Cavinder
Committee Members,	Noah D. Cohen
Head of Department,	H. Russell Cross

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ABSTRACT

Effect of Concentrate Form on Gastric Ulcer Syndrome in Horses.

(December 2011)

Lindsey Rae Huth, B.S., Texas A&M University

Co-Chairs of Advisory Committee: Dr. Dennis H. Sigler
Dr. Clay A Cavinder

Equine gastric ulcer syndrome (EGUS) is common amongst equine athletes of various disciplines and linked to decreased performance. Prevalence among racehorses has been reported to be over 90%, performance horses at 60%, and endurances horses at about 70%. In swine, concentrate form and smaller particle size increase gastric ulceration; thus, the objective of this study was to investigate the effect of concentrate type on EGUS. Quarter Horse yearlings (n=19; 12-18 mo) were blocked by initial EGUS score on a scale of 0 to 4 (0= no ulceration or hyperkeratosis, 4= extensive, deep ulceration) and sex, and utilized in a 77-d cross-over design with two 28-d periods separated by a 21-d washout period. During the first 28-d period, horses were separated into 1 of 2 treatment groups that were all fed Bermuda grass hay and either a commercially available pelleted or textured concentrate. After the initial 28-d period, horses were all fed pelleted feed and Bermuda grass hay for a 21-d washout period then treatment groups were switched for the final 28-d period. Baseline EGUS scores were not different between horses assigned to either treatment (mean 1.1); however, upon treatment, horses fed textured feed acquired a reduced incidence of ulceration as compared to those fed pelleted (mean score of 1.6 vs 1.1, respectively; P =0.02). Degree

and incidence of ulceration was influenced by concentrate form; yearlings fed pelleted feed had higher ulcer scores then those fed textured feed. Therefore, the findings of this study suggests that textured feed may be a effective management tool to aid in the reduction of severity in horses afflicted with EGUS.

DEDICATION

To Linda and Russell, I am blessed to have you as parents and friends. Through the way you live your lives you have shown me that with a strong work ethic and sheer determination, you can achieve anything. It is easy to say that I would not have already achieved so much without your love and support, I love and thank you.

Brandon, you have taught me so much and I do not even think you realize it. When I was a child you taught me how to stand up for myself and not let anyone (or anything) push me around. Also, I have always admired and appreciated your decision to serve our country and now your community with your chosen profession. Always remember that I have the utmost respect and love for you.

To my wonderful husband, you have not merely tolerated my decision to be a professional student, but have supported me in so many ways through this journey. Without your help and devotion I would not have been able to do this and I will always appreciate and value that.

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CHAPTER I

INTRODUCTION

Equine gastric ulcer syndrome (EGUS) is a term used to describe lesions that occur in 2 areas of the equine stomach: the glandular region which primarily affects neonates and foals, and the non-glandular, squamous epithelium which occurs in adults and is the predominant presentation of the disease (Bell et al., 2007a). Hydrochloric acid (HCl), volatile fatty acids (VFAs), and bile acids have been shown to cause damage to the non-glandular region of the stomach and numerous risk factors have been identified and investigated as to their effect on the above mentioned acids' pathogenicity (Murray et al., 1996; Berschneider at al., 1999; Buchanan and Andrews, 2003; Nadeau et al., 2003 Andrews, et al., 2006).

Equine gastric ulcer syndrome is common amongst equine athletes of various disciplines. Prevalence among racehorses actively training has been reported to be as high as 100% with a more accurate rate being between 80% to >90% (Hammond et al., 1986; Murray et al., 1996; Bell et al., 2007b). Show horses and those competing in endurance racing have also been shown to be affected by EGUS with 60% of performance horses (McClure et al., 1999) and 67% of endurance horses (Nieto et al., 2004) having gastric lesions present upon endoscopic examination. Equine gastric ulcer syndrome has been suspected as a cause of decreased performance when no other

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This thesis follows the format of Journal of Animal Science.

could be found and Franklin et al. (2008) provide scientific evidence to support the speculation. Over a period of approximately 2 yr, 100 horses were referred to the Equine Sports Medicine Centre at the University of Bristol due to poor performance. A complete examination was performed and in 4 of the horses, the only clinically significant finding was gastric ulceration. All 4 horses were currently being worked, 1 moderately and 3 intensely, and time since their last race varied from 22 d to 7 mo. All horses were treated with 4 mg/kg BW of omeprazole (GastroGard®) once daily for 4 wks. One of the horses also had substantial glandular ulceration and was concurrently treated with sucralfate at a dose of 20 mg/kg BW every 8 h for 2 wk. After the initial treatment, horses were placed on a maintenance dose of 1 mg/kg BW once/d while in training. Additionally, in 3 of the 4 horses, management changes were implemented including daily access to pasture and *ad libitum* access to forage (the last horse was already being kept at pasture). After treatment, all horses showed an improvement in their performance with all winning or placing in their next races.

A decline in performance may have a negative economic impact on horse owners and trainers alike. The National Cutting Horse Association and the National Reining Horse Association Futurities for 3-year old horses pay out several million dollars a year in prize money; the winners of these 2 events take home $250,000 and $125,000, respectively. In the racing industry, a guaranteed purse of $1 million is at stake in the All-American Futurity for 2-year old horses. These events, and others like them, increase the value of young equine athletes due to their possible earning potential and less than optimal performance will likely result in an economic loss to the owner and the

trainer. The decrease in performance can arise from various symptoms of EGUS; for example, lethargy, pain, behavioral changes, and unwillingness to work. Aside from the immediate economic loss associated with a decrease in performance, when considering breeding stock their value may decrease with lack of performance on the track or in the ring. Lastly, the only FDA approved treatment for EGUS is GastroGard® (http://www.allivet.com/GastroGard-Rx-p/10042.htm) and can cost owners $30 to $40 per d. The recommended treatment length is 28 d resulting in treatment being cost-prohibitive for some owners. This cost does not include those for veterinary diagnosis. With such widespread prevalence of EGUS, and its economic impact on owners, further research on the subject and its prevention is warranted.

CHAPTER II

LITERATURE REVIEW

Anatomy and Physiology

The anatomy and physiology of the equine stomach puts the horse at a high risk of developing gastric ulcers. The equine stomach is divided into 2 regions that are separated by the margo plicatus; the proximal third of the stomach consists of a non-glandular, stratified squamous epithelial region that is considered an extension of the esophagus, and the distal two thirds are covered by glandular mucosa that secretes bicarbonate, HCl, mucus and pepsinogen (Figure 1)(Buchanan and Andrews, 2003; Bell et al., 2007a). The pH varies by region, being near neutral in the esophageal region (cardia) and as low as 1.5 in the pyloric region (Murray et al., 1989). The majority of gastric ulcers occur along the margo plicatus in the non-glandular region accounting for approximately 80% of all gastric ulcers (Murray et al., 1989; Murray 1999; Vatistas et al., 1999; Buchanan and Andrews, 2003). This would be expected due to the frequent contact of the margo plicatus with the acidic gastric fluid; additionally, the lesser curvature is thought to be more affected due to increased contact with the gastric acid (Murray, 1999; Picavet, 2002).

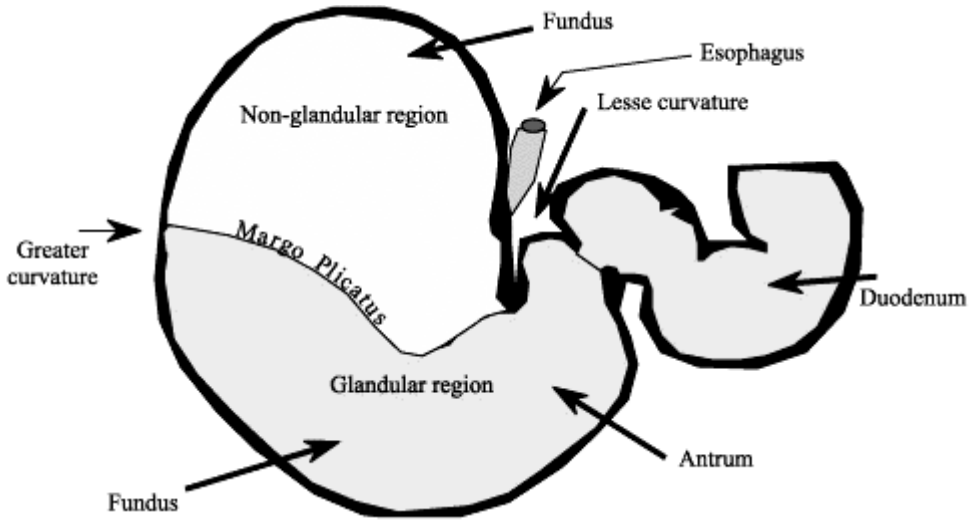

Figure 1. Anatomy of the equine stomach (Dearo et al., 1999)

The little protection that is provided to the non-glandular portion of the stomach arises from the layered nature of the epithelium: stratum corneum (SC), stratum transitionale (ST), stratum spinosum (SS), and stratum basale (SB) which are arranged from the lumen of the stomach to the deepest tissue layer respectively (Argenzio, 1999; Bell et al., 2007a). The regions differ in the mechanisms by which they offer minimal protection: the SC provides a barrier to the diffusion of strong electrolytes such as HCl or Na and the ST and SS contain Na-K ATPase which is involved in transcellular Na transport (Schnorr et al., 1971; Argenzio, 1999). Cell germination occurs at the deepest layer (SB) and proceeds through the cornification process thus providing new, healthy, replacement cells for those that are damaged. Because of the lack of protective buffering mechanisms of the non-glandular portion, contact with low pH gastric fluids cause cellular damage, necrosis and eventual ulceration of the epithelium; however, if exposure to acidic gastric fluid can be eliminated ulcers will spontaneously heal (Murray et al., 2001).

Ulceration of the glandular epithelium is caused by a defect in 1 or more of the intrinsic protective properties present such as lack of proper secretion of bicarbonate or mucus and inhibition or mucosal blood flow (Murray, 1999). When considering the more extensive protective mechanisms of the glandular region, the differences in tissue type may be responsible for the lower incidence and severity of ulceration (Begg & O'Sullivan, 2003). Acidic molecules can readily diffuse into the lumen of the stomach from the mucosa cells via one way transport; therefore, the backflow of these molecules is prevented and the glandular cells are protected (Andrews & Nadeau, 1999; Merrit, 2003). Another protective mechanism in this area is mucosal blood flow; which provides nutrients to the area that promote healing (Murray, 1999; Wallace, 2001). Due to the anatomy and physiology of the glandular region, ulcers in this area heal rapidly and are more common in foals with the cause being a stressor such as disease (Murray, 1999). In adult horses ulcers in this area are associated with non-steroidal anti-inflammatory drug (NSAID) administration presumably due to a decrease in prostaglandin E_2 which is necessary to sustain blood flow and secretion of bicarbonate and mucus (Wallace, 2001; Andrews et al., 2005). This interference with prostaglandin E_2 results in a decrease in pH in the area.

Ulceration in both stomach regions is considered to be caused by an imbalance in the intrinsic protective and aggressive physiologic processes in the stomach (Table 1) (Buchanan and Andrews, 2003). When investigating causes and possible treatment of EGUS, the anatomy and physiology of the equine stomach must be considered in order to understand why a proposed cause or treatment may elicit the hypothesized response.

Table 1. Aggressive and protective mechanisms of the equine stomach

Clinical signs in adults	Clinical signs in foals	Risk factors
Acute colic	Diarrhea	Stress
Chronic colic	Abdominal pain	Transportation
Excessive recumbency	Restlessness	High-concentrate diets
Poor body condition	Rolling	Stall confinement
Anorexia	Dorsal recumbency	Feed deprivation
Loss of appetite	Excessive salivation	Intense exercise
Poor performance	Bruxism	Racing
Changes in behavior	Intermittent nursing	Illness
Lethargy	Poor appetite	Non-steroidal anti-inflammatory drug use
Stretching often to urinate		Mangement changes
Chronic diarrhea		

Etiologies

There are various risk factors associated with EGUS and most are management practices; additionally, these risk factors give rise to pathogenic processes. The most common risk factors include: exercise intensity, feeding practices, and NSAID administration (Furr et al., 1994; Murray & Eichorn, 1996; Vatistas et al, 1999; Wallace, 2001; Fiege et al., 2002; Lorenzo-Figueras & Merrit, 2002; Buchanan & Andrews, 2003; Nadeau et al., 2003; Andrews et al., 2006; Franklin et al., 2008). The pathogenicity of these risk factors arises from alteration of the normal physiologic functions of the equine stomach that are naturally protective.

Volatile fatty acids have been shown to cause ulceration in pigs by damaging the squamous epithelium in an acidic environment (Andrews & Nadeau, 1999) and because horses are fed high concentrate diets like pigs, it is thought the same results are possible. Studies have been done in the horse to determine the effect of various VFAs on the

epithelial integrity of the horse's stomach via the level of barrier functions. Acetic, propionic, butyric and valeric acid at various pH levels yielded different results; acetic acid, in the presence of HCl, was the most pathogenic at a pH < 4.0 whereas valeric acid caused damage at a pH of ≤ 7.0. The barrier function of the stomach mucosa is jeopardized in the presence of these VFAs at varying concentrations and pH levels due to cellular swelling, increased cell permeability and interference with Na transport (Nadeau et al., 2003; Andrews et al., 2006). The same studies suggested that chain length of VFAs is important with longer chain VFAs being more ulcerogenic. Of all the VFAs, acetic acid is present in the highest levels in tissue at 92% and is thought to permeate the epithelium of the non-glandular mucosa more easily than the others, thus, it is possibly more problematic (Andrews et al., 2006).

Volatile fatty acids are important in the pathology EGUS, but hydrochloric acid is considered the main culprit. Sodium transport is imperative to tissue health and when it is altered cell damage occurs. Cell permeability is affected prior to a decrease in Na transport which suggests that the increase in H ions, due to an increase in HCl, causes acidification of the cell layers resulting in a decrease of Na transport. This in turn causes the cells to become turgid and eventually lyse. Apoptosis begins upon the irreversible damage that is done to the cell and this process will eventually lead to ulceration (Argenzio & Eiseman, 1996). Also, HCl activates pepsinogen and the resulting product, pepsin, has been implicated in EGUS (Widenhouse et al., 2002). The combination of an increased amount of H^+ and the reduction in pH lends to HCl having a multifaceted effect on EGUS.

Exercise is one of the primary causes of EGUS among horses of various disciplines. Race horses, western performance, endurance, working ranch and even lesson horses are commonly afflicted with EGUS. One explanation is mechanical: as the horse moves and works, the intra-abdominal pressure increases causing the acidic fluid contents of the distal stomach to be pushed forward and come into contact with the proximal, highly susceptible, non-glandular mucosa. As the exercise duration increases, so does the exposure time of the non-glandular epithelium to the acidic fluid, and ulceration worsens. This could explain why as the exercise duration and intensity increase, so does the severity of ulceration (Lorenzo-Figueras and Merrit, 2002). Furr et al. (1994) found that serum gastrin concentrations increase in exercising horses and it is possible that an increase in gastrin may stimulate HCl secretion and thus lower stomach pH. Other studies have successfully linked exercise to ulceration and attribute both exercise and the management associated with training regimens, such as feeding practices and stall confinement, to ulceration (Hammond et al., 1986; Murray et al., 1996; McClure et al., 1999; Murray, 1999; Vatistas et al., 1999; Bell et al., 2007a; Bell et al., 2007b). Expectedly, horses that are actively, and intensely, training, are at a higher risk of developing EGUS.

The manner in which horses are fed and housed has an effect on EGUS. Most horses in competition or training are housed in stalls with limited or no access to pasture, are placed on high concentrate feeds which limit the amount of forage intake. Furthermore, horses experience periods of feed-deprivation due to feeding schedules. Research has shown that horses kept on pasture have lower rates of ulceration

(Buchanan and Andrews, 2003). Saliva production is stimulated during forage consumption; therefore, the protective benefit of free grazing is that saliva can be produced continuously just as HCl is produced in the stomach, thus preventing an imbalance in the aggressive and protective mechanisms in the equine stomach (Table 1)(Buchanan and Andrews, 2003; Bell et al., 2007a). Additionally, most horses in training or competition are fed high concentrate diets of large volumes and at intervals of approximately 10 to 12 h. The resulting problem is 2-fold: concentrate feeds are high in hydrolyzable carbohydrates which lead to an increase in VFA concentrations and feed deprivation has been shown to induce ulceration (Murray & Eichorn, 1996; Vatistas et al., 1999; Nadeau et al., 2003; Andrews et al., 2006).

There are other risk factors that have been investigated as contributors to ulceration in horses such as transportation and reflux of bile salts (McClure et al., 1999; McClure et al., 2005). Transportation is most likely not the direct stressor, but the feed and water deprivation that horses experience during transportation is probably the cause of ulceration (Buchanan and Andrews, 2003). Bile salts have been suggested to cause glandular ulcers in the pyloric region. The natural occurrence of reflux of bile salts from the duodenum into the stomach would mean that the pylorus is exposed to large amounts of bile acids and result in extensive ulceration of the pylorus. However, this is not considered to be the case because ulcers seen at the pylorus are less severe than those found in the non-glandular region lending to the thought that bile acids do not play a significant role in EGUS (Begg & O'Sullivan, 2003).

The administration of NSAIDs in the adult horse is primarily associated with glandular ulcers as opposed to non-glandular. These drugs interfere with the production of prostaglandin E_2 which is necessary for bicarbonate production and adequate mucosal blood flow which compromises the protective capabilities of the glandular mucosa (Andrews and Nadeau, 1999; Buchanan and Andrews, 2003).

There have been a few studies that investigated the effect of various diet changes on ulcer score. Hayes (2009) investigated the effect of trace mineral supplementation on EGUS score among exercising yearlings, but found no effect. As previously mentioned forage intake stimulates saliva production and is beneficial in raising the pH of the stomach. Additionally, forage type has been investigated to determine if it may result in an effect on the incidence of EGUS. Horses that were on an alfalfa hay and grain diet had higher stomach pH and decreased severity of ulceration when compared to horses on grass hay and grain (Lybertt et al., 2007). Also, horses on alfalfa-grain diets versus those on grass hay diets saw the same effect; the increased Ca levels in alfalfa hay is suspected as the cause of these results (Nadeau et al., 2000). With the ability of diet to change the stomach environment, and subsequently impact ulceration, research regarding the effect of concentrate form could hold potential for better management of EGUS.

Given the numerous risk factors associated with EGUS, it is difficult to identify a single one cause of ulceration in most cases. Instead, it is most often a combination of several risk factors working synergistically that contribute to ulceration. Identifying

possible etiologies and understanding the mechanisms by which they lead to ulceration is imperative to further research in diagnostics, preventions and treatments.

Diagnosis

Currently, the only diagnostic technique available for antemortem detection and monitoring of gastric ulceration is via endoscopic examination. The process of endoscopic examination involves sedating the horse, passing an endoscope into the esophagus and into the stomach in order to visualize the non-glandular and glandular portions of the stomach. Endoscopy is reliable, precise and fairly easy to perform; however, it is not always performed because of limited availability of equipment and cost for the procedure (Andrews et al. 1999).

Andrews and colleagues (2002) investigated the ability of practitioners to accurately classify ulcer severity using various scoring systems by comparing antemortem scores to those assigned upon gross and histological observation during necropsy. When using the Practitioner's simplified (PS) scoring system, which is based on a scale of 0 to 3 (Andrews et al., 1999), there was no significant difference between endoscopic and necropsy examinations. Additionally, when using other scoring systems that attempted to estimate ulcer depth, scores were underestimated when assigned using endoscopic examination versus necropsy examination (Andrews et al., 1999).

Endoscopic examination is still considered an accurate method to diagnose and monitor EGUS, but non-invasive diagnostics have been explored. Measures of sucrose in urine and blood are thought to be potential diagnostic tools to detect EGUS. Normally, if the gastric epithelium is intact, sucrose cannot permeate due to its large

size; therefore, increases in blood or urine concentrations of sucrose may be able to indicate a defect in the gastric epithelium O'Conner et al. (2004), studied the ability of measuring urine concentration of sucrose to determine EGUS in horses with induced ulceration. Upon administration of sucrose via nasoesophageal tube, urine samples were collected at 2 h and 4 h through a urinary catheter that was previously placed. After the 4 h sample, endoscopic examination was performed and results indicated that urine sucrose levels were significantly higher in horses with ulcer severity scores > 1. The ability of sucrose permeability testing was found to have a sensitivity and specificity in detecting ulcer scores > 1 of 83% and 90%, respectively. The information from this study indicates that the method of urine sucrose concentration is reliable, but somewhat impractical due to the labor intensiveness of urine collection. The less invasive and less laborious method of drawing post-sucrose administration blood samples may provide an alternate, practical diagnostic tool for the detection of EGUS. In a recent study, horses were administered sucrose in the manner previously described, then, blood was drawn from a previously placed catheter at 15, 30, 45, 60, and 90 min. For horses with ulcer scores of 1 to 3, peak serum sucrose concentration was seen at 45 min and significantly correlated with ulcer severity (Hewetson et al., 2006). These methods cannot currently replace endoscopy, but would be a valuable tool in aiding in the diagnosis of EGUS.

Treatment

When compared to human disease, EGUS is more closely related to gastro-oesophageal reflux disease syndrome (GERDS) than peptic ulcers (humans do not have a non-glandular portion of the stomach). Like the equine stomach, the squamos gastro-

oesophageal junction in man does not have the inherent protective mechanisms to guard against acid damage (Andrews and Nadeau, 1999). Therefore, treatment protocols for EGUS have been modeled after treatments used for GERDS.

Treatments are aimed at increasing stomach pH > 4.0 in order to create an environment where ulcers can heal. Studies have shown that in a stomach with a pH > 4.0, acidic damage can be reversed (Andrews et al., 2006). Given the dynamic nature of the stomach, healing can vary greatly depending on location, depth, severity, and stomach environment. Ulcers have been shown to begin healing within 24 h of mucosal damage and be completed in 7 d for smaller lesions to over 3 mo (Murray et al., 2001, Bell et al., 2007a). One major difference between EGUS and GERDS is that a bacterial infection caused by *Heliobacter pylori* has been associated with the human form of the disease, whereas, *Heliobacter spp*. have not been identified as a cause of EGUS (Collier and Stoneham, 1997; Collier, 1999; Bell et al., 2007). Given this information, treatment in the horse focuses on increasing the stomach pH rather than a multifaceted approach including antibiotics as in GERDS.

Currently, the only FDA approved medication for the treatment and prevention of gastric ulcers in horses is omeprazole (GastroGard®) (Buchanan and Andrews, 2003). Omeprazole inhibits acid secretion by irreversibly binding the H^+/K^+ATPase enzyme (the proton pump). When uninhibited, the pump facilitates the exchange of a K^+ for a H^+ in the last step of acid secretion in the gastric parietal cells. Upon the release of the H^+ into the lumen, it combines with Cl^- to form HCl (Merrit, 2003). Due to its ability to inhibit the secretion of acid via the proton pump, omeprazole is an invaluable tool in

treating and preventing gastric ulceration in horses (Merrit, 2003; O'Conner et al., 2004; McClure et al., 2005).

Various other treatments for EGUS have been explored and found to be either ineffective or impractical. Sucralfate is often used as a gastro protectant in dogs and humans in various situations and works by forming a gel-like substance and adheres to ulcerated regions preventing further acid damage (Konturek et al., 1989; Hanson et al., 1997). Ranitidine and cimetidine are both histamine H_2 receptor antagonists and inhibit histamine production. Under normal conditions, histamine binds to H_2 receptors on the parietal cells and stimulates acid production. When H_2 antagonists are administered, they competitively bind to histamine binding sites and suppress acid production. Ranitidine is more potent than Cimetidine, but both drugs have low bioavailability and are required in large, frequent doses to be effective (Murray & Eichorn, 1996).

The cost of efficiently treating and preventing ulceration in horses is expensive, being as high as $35 to $40 per d for GastroGard® or more expensive in some cases. Additionally, as with many long-term medicinal therapies, a negative impact on the patient's health is of concern. The normal, acidic environment of the stomach provides conditions favorable for protein digestion and raising the pH of the stomach for an extended period of time could limit protein digestion in the stomach. Because the stomach is the first place of protein digestion in the horse, interference here could decrease the digestibility of protein in the horse's diet. Also, acid-suppression therapy in humans has led to an increase risk in bacterial and viral infection as well as decreases in bone density and a subsequent risk of osteoporosis and fracture (Laheij et al., 2003; Dial

et al., 2005; Yang et al., 2006). So in addition to the economic need for other anti-ulcerogenic treatments and management options, there is an added health component to the dilemma.

Research in hogs has shown that concentrate form has an effect on gastric ulceration with pelleted feed being more ulcerogenic than a concentrate comprised of larger particle size. It is speculated that the smaller particle size results in a more fluid stomach content that contacts the non-glandular epithelium more than a denser, less fluid content would (Erickson et al., 1980; Ayels, 1996; Amory et al., 2006). Given the similarities of anatomy between the hog and horse stomach, it is reasonable to expect the same results in horses feed a pelleted feed (Kararli, 1995). If research in the horse can offer the same results seen in hogs, this would provide a simple, inexpensive, anti-ulcerogenic management option available to the industry.

CHAPTER III

MATERIALS AND METHODS

Horses

Quarter Horse yearlings (n = 19; 12 to 18 mo) owned by the Texas A&M University Department of Animal Science Horse Center facility in College Station, TX were utilized for this study; of the 19 yearlings, 14 were geldings (337.3 ± 30.4kg) and 5 were fillies (328.3 ± 28.7 kg). All of the horses were raised at the Horse Center facility and had not been transported elsewhere. Prior to their use in this trial, horses were kept in groups of approximately 10 yearlings per pasture and were group fed a 14% CP pelleted concentrate twice per d (Producer's Co-op, Bryan, TX).

Horses were administered vaccines in accordance with guidelines of the American Association of Equine Practitioners and anthelminitics in accordance with the herd health maintenance program at the horse center prior to the start of the trial. Research was conducted in accordance with Institutional Animal Care and Use Committee (IACUC) guidelines of Texas A&M University.

Experiment and Treatment Design

This experiment was designed and analyzed using a randomized, controlled, switchback design, consisting of two 28-d periods separated by a 21-d washout period. Horses were blocked by initial EGUS score (Table 2)(Merrit, 2003; Hayes, 2009) and sex. Horses were then randomly assigned to 1 of 2 treatment groups resulting in 1 group of 10 horses and a second group of 9 horses.

During the first period, group 1 received the of 14% crude protein (CP) pelleted ration and coastal Bermuda (CB) grass hay; while group 2 received a 14% CP textured sweet feed (Producer's Co-operative, Bryan, TX) and CB (Table 3). Both treatment groups received CB from the same source during each period. After period 1, horses were turned out to pasture for 21-d in 2 groups so that equal numbers from each treatment group were in each pasture. During the 21-d washout period, horses were group fed the same pelleted concentrate, had *ad libitum* access to CB grass pasture and hay, as well as water. After the washout period, the horses were brought back to the dry lots and fed the opposite rations during period 2: group 1 received textured feed and group 2 received pelleted feed. Period was defined by feed type to determine the influence on EGUS scores.

Table 2. Modified EGUC scoring system used to identify potential ulceration of yearling horses fed either pelleted or textured ration (Merrit, 2003)

Score	Description
0	Epithelium is intact throughout; no hyeremia or hyperkeratosis
1	Mucosa is intact, but there are areas of hyperemia and hyperkeratosis
2	Small, single or multi-focal, non-bleeding erosions or ulcers
3	Large, single or multi-focal, erosions or ulcers; or any actively bleeding ulcer
4	Extensive ulcers, with areas of deep submucosal penetration

Table 3. Nutrient analysis by feed type (DM)

Nutrient	Period 1 Pelleted	Period 1 Textured	Period 2 Pelleted	Period 2 Textured	CB Hay
Dry Matter %	100	100	100	100	100
Protein %	15.3	15	16.8	17.3	12.5
Net Energy (Mcal/lb)	0.81	0.83	0.81	0.82	0.67
ADF%	11.8	15.6	12.4	11.5	27.5
TDN-based on ADF	82.8	79.3	82.3	83.2	64.7
Calcium, %	0.69	1.04	1.16	0.72	0.5
Phosphorus, %	0.58	0.6	0.61	0.65	0.25
Potassium, %	1.03	1.11	1.09	1.1	1.54
Magnesium, %	0.23	0.29	0.30	0.25	0.18
Sodium, %	0.27	0.37	0.41	0.23	0.07
Zinc, ppm	96	110	101	72	39
Iron, ppm	26	27	33	33	34
Copper, ppm	35	40	37	44	13
Manganese, ppm	87	156	299	78	96

Diet

Horses were individually fed 2.25% body weight (BW) per d; hay and grain were fed in a 1:1 ratio. The total ration was equally divided and fed twice daily. The morning feeding began at 0630 h and the evening feeding began at 1730 h. Horses were allowed 30 min to consume grain and 2.5 h to consume hay. Refusals were collected, weighed, and recorded at the end of the allotted time for each feeding in order to determine intake. To ensure the horses were being fed at 2.25% BW, horses were weighed every 7 d and rations adjusted accordingly.

For horses receiving the textured diet, a period of 5 d was allowed for transition to the new feed type by replacing 25% of the total ration with textured concentrate each day (i.e. day 1 ration was, textured to pelleted, 1:3, day 2 and 3 was 1:1, day 4 was 3:1

and day 5 was all textured). The same process was reversed and followed when switching from the textured diet back to the pelleted diet.

Concentrate and hay samples were analyzed for nutrient content (Texas AgriLife Extension Service – Soil, Water, and Forage Testing Laboratory, College Station, TX). Concentrate samples were taken weekly, pooled within the period, and then analyzed. The hay used for the entire project was from the same source and cut; therefore, core samples were taken from approximately 25 bales at the beginning of the project and analyzed. Samples were placed in zip-loc bags and stored in a freezer until delivery to the testing lab (Table 3).

In both periods horses were fed in individual concrete stalls (3 x 3 m) to ensure they had access only to their assigned treatment. This also allowed investigators to collect any refusals. Water, from the same source, was available *ad libitum* in each feeding stall for each feeding.

Housing

During both treatment periods (initial and switch back) yearlings were housed in 4 adjacent dry lot pens (22.50 x 15.39 m) at the Texas A&M Horse Center. This ensured restriction of nutrient intake to the diets offered during the trial. Horses were grouped so that there was equal representation of the 2 treatment groups among the individual pens. Water tanks were available in each pen and filled during each feeding to ensure *ad libitum* access.

During the 21-d washout period, horses were randomly divided into 2 groups, with equal representation from each treatment group and turned out to pasture. The

pastures were of equal size, contained the same forage and water sources, and were located adjacent to one another with a shared fence.

Exercise

Horses were moderately exercised 3 d/wk for 20 min. The regimen was not strenuous and served to maintain soundness of mind. Exercise consisted of a rotation between use of a mechanical horse walker and free-exercise in a large dry lot. The mechanical horse walker regimen consisted of a 5 min warm up at 1 m/s (walk), 10 min at 2.9 m/s (long trot), and again at 1 m/s for a 5 min cool-down walk. The walker - contained 6 free-stall compartments and equal representation from both treatment groups was achieved. Exercise sessions took place in mid-afternoon (prior to the evening feeding) and horses were on a rotational schedule to ensure that the same horses were not always worked directly before the evening feeding.

Endoscopic Examination

Endoscopic examination of the stomach was performed 4 times throughout the project; d-0 and d-28 for each period (1 and 2). Before each endoscopy, horses were placed into the individual concrete feeding stalls to be fasted for 18 h prior to examination, with water available until 1.5 h prior. Horses were fasted in order to allow for an unobstructed view of both the non-glandular and glandular portions of the stomach so as to assign a more accurate ulcer score. Horses were monitored during this time and any feces removed to guard against copraphagy as fecal matter would interfere with complete visualization of the gastric lining.

Horses were placed into stocks and mildly sedated with 200 to 250 mg of xylazine hydrochloride administered intravenously. A humane twitch was then applied to safely restrain the horses and to facilitate the passage of a lubricated 1-m long, 1.65-cm diameter nasoesophageal tube. The purpose of this tube was to protect the 3-m flexible endoscope (Olympus, Tokyo, Japan) from intrapharyngeal retroflexion. The endoscope was then passed through the esophagus to the stomach which was insufflated upon reaching the cardia to increase the surface area and ensure more complete visualization and accurate scoring. Water was used to rinse any debris from the stomach wall when needed. The non-glandular region was scored, on a scale from 0 to 4, using a modified version of the Equine Gastric Ulcer Council's scoring system (Table 3). The internist was familiar with the scoring system as it had been used in previously by investigators in this laboratory (Lybbert, 2007; Hayes, 2009).

At least 4 relevant pictures of the stomach were taken of each horse at every examination. At the conclusion of the examination, the stomach was deflated by aspiration to prevent discomfort due to gas distension and the horses were returned to dry lots. In cases where the horse seemed heavily sedated, they were returned to the individual stalls until deemed coherent enough to be returned to the dry lots. About 30 min after the last horse was examined, hay was offered as a group in the dry lots. During the evening feeding on endoscopy days, horses were fed one-quarter their normal grain and group fed hay. The next morning horses were fed one-half their normal feed and group fed hay again. The next evening, horses were fed their full normal ration and were fed hay individually if the examination was a pre-period examination. If it was a

post-period examination, horses were group fed hay again the next evening and returned to pasture by 48 h post endoscopic examination.

Statistical Analysis

Data were analyzed as repeated measures using the mixed model procedures of SAS (SAS Institute, version 9.2, Cary, NC, USA). The model included fixed effects of horse, treatment, period and treatment by period interaction. The random effect was horse nested within treatment. Least square means and least square means differences were analyzed using a pairwise t-test to determine if any differences between treatments, periods or treatments and periods were significant. A P-value ≤ 0.05 was considered significant. Table 4 was analyzed using the least significant differences test for differences among treatment means (Snedecor and Cochran, 1974).

Glandular ulcers were disregarded due to their rarity of occurrence in this study. Therefore, the statistical analysis includes only non-glandular ulcer data.

CHAPTER IV

RESULTS

Ulcer Score Data

Yearling horses were subjected to a switchback of treatment groups; therefore, period effect was evaluated and no significant difference was found in the initial score upon the beginning of period 1 or period 2 in either treatment group (Table 4 and Figure 2). However, treatment, pelleted vs textured feed, did have an effect on ulcer score which was evident by the scores by the end of both period 1 and 2 (Table 4 and Figure 2). Upon completion of each treatment, thus the end of each period, EGUS scores were evaluated and a significant effect of period ($P = 0.0006$) and a treatment by period interaction ($P = 0.0004$) was found. During period 1, all horses regardless of treatment experienced an increase in severity of ulceration; however, horses on textured feed had a smaller increase in ulcer severity (1.0 to 1.7; $P = 0.03$) than horses fed the pelleted diet (0.9 to 2.2; $P = 0.0005$)(Table 4 and Figure 2). Upon completion of period 2, both treatment groups saw a decrease in score; pelleted group scores at d-0 were 1.4 and decreased to 1.0 while the textured group scores at d-0 were 1.1 and significantly decreased to 0.5 ($P = 0.39$ and 0.02 respectively)(Table 4 and Figure 2).

Overall, horses that were fed pelleted feed had an increase in score from d-0 to d-28 of 0.4 ($P = 0.02$)(Table 4 and Figure 2). When comparing horses that had been on pelleted feed for 28 d compared to those on textured feed for 28 d, those on the textured diet had scores that were 0.5 lower than those on the pelleted diet ($P = 0.03$)(Table 4 and Figure 2).

Table 4. Mean ulcer scores of yearling horses by endoscopy session and treatment group, ^{a,b,c,d} means sharing similar superscripts do not differ (P< 0.05)

	Endoscopy			
Treatment	**1**	**2**	**3**	**4**
Textured	$1.1^{b,c}$	1.7^{d}	$1.1^{b,c}$	0.5^{a}
Pelleted	$0.9^{a,b,c}$	2.2	$1.4^{b,c,d}$	1.0^{b}

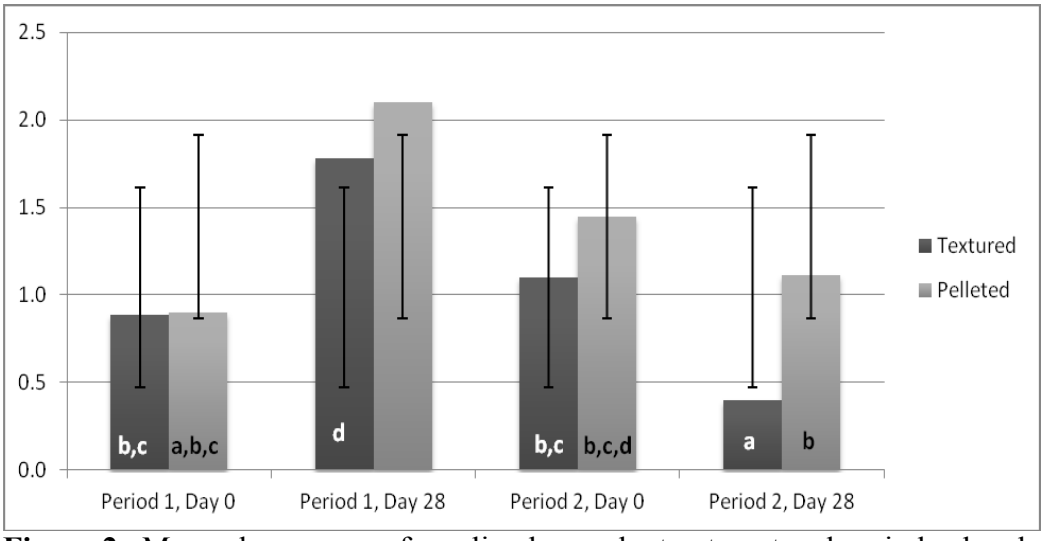

Figure 2. Mean ulcer scores of yearling horses by treatment and period, a,b,c,d means sharing similar characters do not differ (P < 0.05)

For this study it was hypothesized that concentrate in pelleted form would be more ulcerogenic versus textured concentrate. Data analysis was complicated by the fact that score data were not truly continuous, they are ordinal and discrete. Also, there were repeated measures for individual horses that occurred over 2 periods therefore, score was not independent. A pairwise t-test was done in order to determine if there was a significant difference in scores between the treatments in each period. Significant effects were seen for treatment and average scores for pelleted treatment groups were

found to be higher lending support to the hypothesis. Period was highly significant with scores decreasing in both treatment groups in period 2 (P = 0.0006). Scores on d-28 of period 2 were significantly lower than their d-28 of period 1 counterparts; the average for d-28, period 1 of the pelleted group was 1.2 higher than the day 28 score during period 2 (P = 0.0083). Period 1, d-28 scores of the textured group differed by a value of 1.2 (P = 0.01).

Grain Intake Data

Table 5 shows the average daily intake (ADI) for periods 1 and 2 by period and treatment while Tables 6 and 7 show the nutrient intake from grain consumption for periods 1 and 2 respectively.

Table 5. ADI of grain for yearling horses by treatment and period (90 % DM basis)

	Period 1 Pelleted	**Period 1 Textured**	**Period 2 Pelleted**	**Period 2 Textured**
ADI (kg)	3.50	3.52	3.85	3.85
SE	0.06	0.04	0.02	0.02

Table 6. Nutrients consumed from grain for yearling horses by treatment for period 1 (DM basis)

Nutrient	Pelleted (g)	Pelleted (g/kg BW)	Textured (g)	Textured (g/kg BW)
Dry Matter %	90	90	90	90
Protein	535.50	1.54	528	1.52
Net Energy	28.35	0.08	29.216	0.08
ADF	413.00	1.19	549.12	1.58
TDN-based on ADF	2898.00	8.36	2791.36	8.01
Calcium	24.15	0.07	36.608	0.11
Phosphorus	20.30	0.06	21.12	0.06
Potassium	36.05	0.10	39.072	0.11
Magnesium	8.05	0.02	10.208	0.03
Sodium	9.45	0.03	13.024	0.04
Zinc	0.33600	0.00097	0.03872	0.00011
Iron	0.09100	0.00026	0.09504	0.00027
Copper	0.12250	0.00035	0.14080	0.00040
Manganese	0.30450	0.00088	0.54912	0.00158

Table 7. Nutrients consumed from grain for yearling horses by treatment for period 2 (DM basis)

Nutrient	Pelleted (g)	Pelleted (g/kg BW)	Textured (g)	Textured (g/kg BW)
Dry Matter %	90	90	90	90
Protein	646.80	1.70	666.05	1.75
Net Energy	31.19	0.08	31.57	0.08
ADF	477.40	1.26	442.75	1.16
TDN-based on ADF	3168.55	8.33	3203.2	8.43
Calcium	44.66	0.12	27.72	0.07
Phosphorus	23.49	0.06	25.025	0.07
Potassium	41.97	0.11	42.35	0.11
Magnesium	11.55	0.03	9.625	0.03
Sodium	15.79	0.04	8.855	0.02
Zinc	0.38885	0.00102	0.27720	0.00073
Iron	0.12705	0.00033	0.12705	0.00033
Copper	0.14245	0.00037	0.16940	0.00045
Manganese	1.15115	0.00303	0.30030	0.00079

Hay Intake Data

Table 8 shows the average daily intake (ADI) for periods 1 and 2 by period and treatment while Tables 9 and 10 show the nutrient intake from grain consumption for periods 1 and 2 respectively.

Table 8. ADI of hay for yearling horses by treatment and period (90% DM basis)

	Period 1 Pelleted	**Period 1 Textured**	**Period 2 Pelleted**	**Period 2 Textured**
ADI (kg)	3.29	3.43	3.64	3.74
SE	0.03	0.06	0.07	0.05

Table 9. Nutrients consumed by yearling horse from hay by treatment for period 1(DM basis)

Nutrient	**Pelleted (g)**	**Pelleted (g/kg BW)**	**Textured (g)**	**Textured (g/kg BW)**
Dry Matter %	90	90	90	90
Protein	411.25	1.19	428.75	1.23
Net Energy	22.04	0.06	22.981	0.07
ADF	904.75	2.61	943.25	2.71
TDN-based on ADF	2128.63	6.14	2219.21	6.37
Calcium	16.45	0.05	17.15	0.05
Phosphorus	8.23	0.02	8.575	0.02
Potassium	50.67	0.15	52.822	0.15
Magnesium	5.92	0.02	6.174	0.02
Sodium	2.30	0.01	2.401	0.01
Zinc	0.12831	0.00037	0.13377	0.00038
Iron	0.11186	0.00032	0.11662	0.00033
Copper	0.04277	0.00012	0.04459	0.00013
Manganese	0.31584	0.00091	0.32928	0.00095

Table 10. Nutrients consumed by yearling horse from hay by treatment for period 2 (DM basis)

Nutrient	Pelleted (g)	Pelleted (g/kg BW)	Textured (g)	Textured (g/kg BW)
Dry Matter %	90	90	90	90
Protein	455.00	1.20	467.5	1.23
Net Energy	24.39	0.06	25.058	0.07
ADF	1001.00	2.63	1028.5	2.71
TDN-based on ADF	2355.08	6.19	2419.78	6.37
Calcium	18.20	0.05	18.7	0.05
Phosphorus	9.10	0.02	9.35	0.02
Potassium	56.06	0.15	57.596	0.15
Magnesium	6.55	0.02	6.732	0.02
Sodium	2.55	0.01	2.618	0.01
Zinc	0.14196	0.00037	0.14586	0.00038
Iron	0.12376	0.00033	0.12716	0.00033
Copper	0.04732	0.00012	0.04862	0.00013
Manganese	0.34944	0.00092	0.35904	0.00094

There were minimal refusals of hay or grain amongst all horses in both treatment groups throughout the study. Occasional refusals consisted of hay left on the ground or the horse ran out of time; no horse truly refused any grain or hay throughout the study. Also, there were minimal clinical symptoms of ulceration such as mild abdominal discomfort, behavior changes, and eating slower.

CHAPTER V

DISCUSSION

In this study horses that were fed textured concentrate saw, on average, a lower ulcer severity score than horses on the textured concentrate. Ulcer severity was not improved, but the scores of those horses fed textured fed increased by smaller margins than those on pelleted feed. This could be due to a less ulcerogenic environment in the stomach as a result of particle size. In hogs, who share similar anatomic and physiologic characteristics with the equine stomach, ulceration of the pars esophageal region is a problem which can decrease profit for producers. Research in hogs has proven that ulceration severity is increased when particle size is decreased such as in pelleted feeds (Ayles et al., 1996; Amory et al. 2006). One hypothesis is that the smaller particle size creates a more fluid stomach content which results in a sloshing effect; thus, increasing the amount of time the acidic stomach contents comes into contact with the non-glandular region. Considering this information, and the popularity of pelleted feeds in the horse industry, the effect of concentrate form on EGUS could have wide-spread implications in the management of EGUS.

The long-standing consensus among much of the literature is that horses on pasture have lower ulcer scores and that was seen in this supported by the results seen in this study. However, recent studies have seen otherwise. Nadeau et al., 2000 saw that horses on a textured grain and alfalfa diet had lower scores than horse with *ad libitum* access to grass hay (much like a pasture diet). In a second study (Lybbert, 2007), the design used was the same as the one employed for this author's study. When horses

were placed in the pasture for the washout period, all scores increased; this was viewed as an increase from score 2 to score 3. This difference could be explained by individual variances among horses and their reactions to stress. For example, during the washout in both studies (Lybbert, 2007 and the current study) horses were group fed in a pasture setting. Some horses, such as a less dominant one, may have been continually run off from their feed and found this more stressful that being fed alone, in a stall. There are numerous risk factors associated with EGUS and it becomes very difficult to avoid some confounding variables such as various stressors.

Average ulcer scores were lower for both groups of horses in period 2. One possible theory to explain this is the horses were more conditioned to the environment and feeding practices; thus, stress was minimized and ulceration was not exacerbated. During the first period horses were housed in large groups in pastures, a housing situation that has been proven to help decrease the incidence and severity of ulceration (Murray et al., 1996; Vatistas et al., 1999; Buchanan and Andrews, 2003; Bell et al., 2007a). Moving the horses abruptly from a pasture environment with *ad libitum* access to forage, to the environment they were housed in for the study could have caused stress and thus an increase incidence and severity of ulceration. In the first period, scores in both groups increased significantly which would support the theory that the stress of the environment change played a role in ulcer score. Interestingly, the scores of the textured group did not increase to the extent of the scores of the pelleted group; however, this difference was not significant.

It should be noted that no horses in the study had ulcer scores of 4 which would indicate severe ulceration. It would be interesting to see if the textured diet caused a larger reduction in score, a lesser reduction in score or no change at all. Also, because these horses had minimal clinical signs and fairly innocuous endoscopies, it is unclear if any of them would have been candidates for treatment. Since there is no correlation between ulcer severity and clinical symptoms and no clear evidence that there is a point at which performance is hindered, it should be noted that horses in the industry with similar ulcer scores seen in this study may not ever present as potential EGUS cases.

Since there is not a singular cause of EGUS, it should be understood that there is not a singular solution when considering the various management changes that have been implicated in the aid of reducing the severity of the disease. As previously mentioned, horses are individuals and react differently to stress and various situations; therefore, what helps one horse may not help another. This study did provide evidence that horses benefit from a textured diet, and it is an easy management change that most horse owners could make, but it should not be considered a cure or treatment.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Equine gastric ulcer syndrome has been the focus of much previous and current research, but researchers are still left with many questions. This is largely due to the vast range of risk factors associated with EGUS; hence, it is very difficult, if not impossible, to eliminate confounding variables in experimental designs. Horses most likely have been afflicted with EGUS prior to our realization of the disease and as research has shown, the prevalence of EGUS among equine athletes is quite high (Hammond et al., 1986; Murray et al., 1996; McClure et al., 1999: Bell et al., 2007b). Sadly, many horse owners are unable to treat, and prevent, ulceration in horses due to treatment being cost-prohibitive. Currently, the only FDA approved medication (GastroGard®) for treatment and prevention of ulcers costs approximately $39/d. Since treatment is recommended for 28 consecutive days, this would reach amounts nearing $1200 to treat 1 horse and this does not include fees incurred for diagnostics and veterinary services. The high cost of treating such a commonly occurring disease makes finding alternative treatments very attractive.

In this study the property, or properties, of the concentrate form that may be causing the observed results was not investigated. The experiment was designed to simply determine if there may be an effect of concentrate form on severity of ulceration. The results seen in this study hold some promise as ulcer scores were lower for horses on the textured diet when compared to the pelleted diet. Although textured feed did not seem to improve ulceration, it did seem to offer some protection to the stomach. When

ulcer scores went up in both groups during period 1, the textured group average score increased by a smaller margin than the average of the pelleted group suggesting that textured feed may be less ulcerogenic. At this time it does not seem that concentrate form can be used as a treatment for healing EGUS, but it may be beneficial to feed horses textured feed that are being treated for ulcers or may be more prone to ulceration. Further research avenues should include the ability of textured feed to maintain ulcer-free horses, its effect when fed with alfalfa, and the mechanism by which it may offer protection.

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